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TITLE PAGE

TITLE: Efficacy of Sleep Position Modification to Treat Positional Obstructive Sleep Apnea

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HIGHLIGHTS

- Well-tolerated, inexpensive therapies are needed to treat obstructive sleep apnea (OSA).
- Supine-dependant OSA is defined as AHI supine/2 ≥ AHI non-supine
- We assessed a novel device to prevent supine sleep in a placebo-controlled trial
- The device was well-tolerated,with a significant reduction in supine sleep and AHI
- Benefits were greatest in those with baseline AHI>20.

ABSTRACT

Objective/Background: To assess the feasibility and efficacy of sleep position modification in preventing supine sleep and improving sleep-disordered breathing and relevant clinical outcomes in positional Obstructive Sleep Apnea (OSA) patients.

Patients/Methods: Eighty-six consecutive participants with moderate positional OSA on routine diagnostic polysomnography underwent a randomized controlled parallel group design trial of 4-weeks treatment using a sleep position modification device (active) or sleep hygiene advice (control). Outcomes were measured at baseline and following a 4-week treatment period.

Results: There was a significant reduction in the amount of supine sleep in the active group (mean±SD change from baseline, active group 99.5±85.2 minutes, control group 68.6±103.2 minutes, p=0.002) and an improvement in apnea-hypopnea index (AHI) (active group reduced by 9.9±11.6, control group reduced by 5.3±13.9, p=0.013). Post-hoc analyses indicated that positional therapy was most effective for patients with baseline AHI cut-off above 20 (p=0.02). Logistic regression showed that a treatment response (AHI<10) was more likely in the active group (OR=5.57), and those with higher baseline nadir oxygen desaturation (OR=1.95) and non-supine AHI (OR=0.52). There were no significant improvements in quality of life, daytime sleepiness, mood, symptoms, neuropsychological measures or blood pressure in the active group.

Conclusions: The position device utilized in this study was effective in reducing supine sleep and AHI, which was significant in those with baseline AHI ≥20. Longer duration studies of physical treatments that modify sleep position are needed to explore further whether additional clinical benefits in are achievable.

Keywords: Obstructive Sleep Apnea, positional obstructive sleep apnea, non-CPAP treatment of sleep apnea, neuropsychological function

1. INTRODUCTION

Obstructive sleep apnea (OSA) is a common disease, estimated to affect 4% of men and 2% of women in the 30-60 year old age group.[1] The two main treatments for OSA (continuous positive airways pressure (CPAP) and mandibular advancement devices) are effective, but adherence and cost remain an issue for many patients. In particular, patients from lower socioeconomic backgrounds are less receptive to CPAP treatment for their OSA.[2] There is a clear need for well-tolerated, inexpensive and simple treatments, particularly for those with less severe disease.

Positional OSA is reported to be present in between 50%-60% of all patients with OSA, [3] arbitrarily defined as a supine apnea-hypopnea index (AHI) at least twice that of the non-supine AHI.[3,4] A number of mechanisms have been proposed to explain this observation. These include a posture-dependant structural change in the upper airway, elevation of the diaphragm in the supine position and consequent increased upper airway collapsibility or reduction in upper airway muscle activity with the change from lateral to supine sleep position.[3,4] Several studies in anaesthetized normal human participants have shown an increase in upper airway calibre in the lateral recumbent position compared to supine,[5-7] suggesting that passive anatomic changes are involved. A recent study of awake OSA and healthy participants has shown a change in shape but not size of the velopharyngeal and oropharyngeal airway from the supine recumbent to lateral position during wakefulness, and smaller velopharyngeal cross-sectional area of OSA compared to control participants in the supine but not lateral recumbent positions.[8] During sleep, upper airway closing pressure did not differ in

the supine compared to the lateral position, but opening pressure was significantly reduced in the lateral position. [9]

Patients with positional OSA are likely to be younger, less obese, have less severe OSA and less objective daytime sleepiness on Multiple Sleep Latency Test compared to those with non-positional OSA.[3,10] Given the issues with adherence with CPAP treatment, particularly in this population of patients, alternative treatments for avoiding supine sleep have been developed and tested in research studies. One of the most widely reported methods in the literature on positional therapy,[11] the so-called tennis ball technique, involves attaching on of more pockets to the back of a shirt that is worn during sleep. Tennis balls are then placed in the pockets along the spine, thus preventing the patient from sleeping in the supine position. Several studies have investigated the therapeutic benefits of avoiding supine sleep using the tennis ball technique and other devices that prevent supine sleep, as well as alarm systems that awaken patients when they are in supine sleep.[12-18] The benefits shown for sleep disordered breathing are inconsistent across studies and the impact on daytime function has not been adequately investigated. Only a few randomized controlled trials comparing positional devices with continuous positive airways pressure (CPAP) have been conducted over the last 20 years.[19] Two of these studies were relatively small (N<20), and found that the improvements in AHI and sleep hypoxemia after positional therapy (soft ball back pack and a thoracic anti-supine band) were significantly less than the CPAP benefit.[20, 21]No significant difference in neurobehavioral outcomes or sleepiness was observed, possibly due to insufficient sample size. More recently, newer therapies have been developed including a novel body position orienting device (BuzzPOD, Gorman ProMed Pty Ltd) and a neck-worn vibrating device, both of which reduced supine sleep and have demonstrated a statistically significant improvement in AHI, however no measures of daytime function were recorded.[22,23]

Positional therapy appears to be a promising treatment option for a large number of OSA patients, however many studies have been conducted on a comparatively small number of participants and there is an urgent need for further controlled studies to examine its efficacy. The current study aimed to determine whether sleep position modification is efficacious in preventing supine sleep, and evaluate improvements in sleep disordered breathing and clinical outcomes using a large randomised controlled study.

2. METHODS

A randomized, conservative treatment controlled, parallel group trial over 4 weeks was carried out to assess the efficacy of a sleep position modification device in treating patients with positional OSA. The study was carried out in the Institute for Breathing and Sleep at Austin Health, Melbourne, Australia. This study was approved by the Austin Health Human Research Ethics Committee, and informed written consent was obtained from all participants.

2.1 Participants

One hundred and sixteen eligible OSA patients from Austin Health were approached following diagnostic PSG, of whom 86 agreed to participate (Figure 1). The reason given for non-participation in all cases was the time commitment involved. Inclusion criteria were: at least 18 years of age, supine OSA (supine AHI at least twice the non-supine AHI) on overnight diagnostic PSG, total AHI $\geq$ 10, and at least 4 hours of sleep with at least 30 minutes sleep in both the lateral and supine recumbent positions and 30 minutes of REM sleep. Patients with minimum blood oxygen saturation less than 75% in REM or 80% in non-REM were excluded, as were patients with clinically significant co-existing disease (e.g. diabetes, unstable ischemic heart disease) or sleepiness deemed to be unsafe and requiring urgent treatment (e.g. history of falling asleep while driving or working, or an Epworth Sleepiness Scale (ESS)[24] score greater than 16. Participants were also excluded if they had any musculo-skeletal condition that precluded moderate exercise (as this was part of the

sleep hygiene instructions) or lying on their side while asleep. To ensure valid interpretation of the neurobehavioral tests, subjects were required to be fluent in the English language and have no history of cerebrovascular disease, closed head injury associated with loss of consciousness greater than 15 minutes duration, psychiatric illness, or alcohol or drug abuse.

2.2 Study Measures

2.2.1 Polysomnography

All participants underwent standard in-laboratory PSG at baseline and after treatment, as previously described, using the S-series (Compumedics, Melbourne, Australia) sleep monitoring system.[25] The analysis of the PSGs were performed blinded to the treatment arm.

2.2.2 Questionnaires

In order to examine sleepiness, daytime impairments and mood, participants completed the ESS, Functional Outcomes of Sleep Questionnaire (FOSQ),[26] Symptoms of Sleep Apnea Questionnaire (SASQ),[27] and the Profile of Mood States at baseline and follow-up.

2.2.3 Neuropsychological test battery

Participants completed a neuropsychological test battery, which was designed to measure vigilance (Psychomotor Vigilance Task),[28] response inhibition (Stroop[29]), information processing (Trail Making Task A and B[30], digit symbol substitution task[31]), working memory (digits forwards and backwards[27]) and verbal fluency (Controlled Oral Word Association Task[32]) at baseline and follow-up.

2.2.4 Blood pressure and anthropometric assessment

The mean of 3 resting seated blood pressure measurements, weight, height, neck circumference, waist and hip girth were recorded at two timepoints, baseline and follow-up.

2.3 Procedures

Potential participants attended a screening session to provide written informed consent and to assess their eligibility. Eligible participants completed baseline assessment and were then randomized to 4 weeks treatment with either a "Ten Point Guide to Improving Your Sleep Apnea with Healthy Lifestyle Changes" (Appendix 1) which included suggestions for exercise, weight loss and sleep in the lateral position (control group), or the same information plus a tennis ball sleep position modification device (active group). The Guide was a written trifold brochure that was given to each participant by the researcher and each point was discussed. The sleep position modification device (see photograph) is a band of stretch cotton worn around the chest, just below the nipple line and with straps over the shoulder to hold it in place. The band was secured at the front with buttons and the ball was contained in a pocket at the rear, over the thoracic spine. A randomisation sequence at a 1:1 active:control ratio was computer generated by a third party and the investigators were provided with sealed envelopes. These were opened in order of enrolment. Participants in the active group completed a daily diary to record nightly hours of use of the position device. All participants in the active group self-reported using the device for at least 6 hours every night. The purpose and design of the study was concealed to the control participants. These lifestyle recommendations are an integral part of usual clinical care of OSA patients. At the end of the 4-week treatment period, participants had a repeat sleep study in the laboratory (the active group wore the positional device on that night) and completed follow-up assessments. After the study, participants were referred back to their treating sleep physician.

2.4 Statistics

Statistical analysis was performed using SPSS V17.0, 2008 (SPSS Inc., IMB; Chicago, IL), and was performed blinded to the treatment condition. Initial power and sample sizes were calculated[33] with minutes of supine sleep as the primary outcome variable, using data from 52 concomitant Sleep

Laboratory patients who had diagnostic sleep studies in our sleep laboratory which showed positional OSA. These participants had an average total sleep time of 314.0 ± 77.0 minutes and 118.8 ± 82.8 minutes of supine sleep. With a 50% reduction in supine sleep being taken as clinically meaningful, for a power of 90% and α=5% the total sample size required was 66 (N = 33 in each group). Additional participants were recruited to account for attrition.

Repeated measures 2-way ANOVA was used to compare treatment responses (AHI, supine sleep, oxygen saturations and performance outcomes) according to group (control vs active). ANCOVA was performed when a significant correlation was observed between a baseline sleep parameter and the change in outcome variable(s).[34] A chi-square statistic was calculated to measure the likelihood of participant achievement of a complete response according to group allocation. A complete AHI response was defined *a priori* as a reduction in the AHI to less than 10; a partial response was defined as a reduction of at least 50% in the AHI, but still > 10, plus a significant improvement in symptoms. Independent t-tests were performed to identify baseline measures in which there were differences between responders and non-responders (i.e. those who had post-treatment AHI < 10 and those with post-treatment AHI ≥ 10 respectively). Those variables that differed at the level of $p \leq 0.1$ were then entered as covariates into a binary logistic regression model. Because the ANCOVA showed a significant interaction between group (active vs. control) and AHI at baseline, the interaction factor ($AHI_{baseline}$ x group) was entered into a second model. The backwards stepwise method was used with AHI reduction to < 10 (i.e. responder) as the categorical dependant variable. For all continuous variables, odd ratios were standardized to reflect a change of 1 standard deviation in each variable. All data are reported as mean ± SD.

3. RESULTS

3.1 Participants

Forty seven participants were randomly allocated to the active group (78.7% male, age 48.0 ± 11.2), and 39 were randomized to the control group (76.9% male; age 51.2 ± 11.4). Three participants failed to complete the active arm and 2 participants failed to complete the control arm.

Participants had moderate OSA (AHI 20.9 ± 9.4), were middle-aged (49.5 ± 11.4 years), 79% were overweight or obese (BMI range 19.9 – 60.0kg/m^2) and predominantly males (78%) (Table 1). Subjectively, they had mild daytime sleepiness (ESS 10.0 ± 5.2), impaired quality of life (FOSQ total score 3.3 ± 0.5), and elevated symptom levels (SASQ 58. 5 ± 1 9.9) compared to reported normal values.[26]

3.2 Sleep Position

Participants spent 143.5 ± 87.0 minutes supine in their baseline sleep study, which was nearly half of total sleep time. The amount of supine sleep fell significantly in both groups (Table 1, Figure 2A), however position modification was significantly more effective than sleep hygiene advice alone ($p = 0.001$).

3.3 Sleep Disordered Breathing

The total AHI was significantly reduced in both groups (Figure 2B). Potential interaction with all baseline variables (PSG and demographics) and improvement in AHI was explored. There was no significant correlation between change in AHI and baseline AHI for the group as a whole. The change in AHI correlated significantly with baseline AHI in the active group (Spearman's rank correlation $R = -0.56$, $p < 0.001$) but not in the control group ($R = -0.16$, $p = 0.33$). To allow for this, a univariate analysis of variance was done between AHI post treatment and group, with $AHI_{baseline}$ as covariate (ANCOVA).[26] This showed a significant effect of both $AHI_{baseline}$ ($F = 5.43$, $p = 0.022$) and group ($F = 6.49$, $p = 0.013$) on post treatment AHI.

Figure 3 shows the individual change in AHI from baseline to post-treatment for each participant in the active and control groups separately. There was a fall in AHI to < 10 in 67.6% of participants treated with the postural device and in 48.4% of control group participants, but this was not statistically significant (χ^2 = 2.48, p = 0.1). A further 15% of active treatment group and 18% control group participants had a partial response.

Exploratory post-hoc analyses were conducted to determine whether there is a baseline AHI cut-off above which a positional device is effective. Using a cut-off of moderate to severe baseline OSA (AHI ≥ 20) (25 active and 23 control group participants), the 2-way ANOVA showed a significant treatment benefit for those randomized to the active treatment group (p = 0.028); that is, for those with baseline AHI ≥20, there was a significant treatment response (AHI < 10) to the position device.

There was a significant but equivalent fall in the arousal indices in both groups, but no significant difference between active and control groups post treatment (p = 0.08). Sleep oxygenation improved significantly with the position device but was not statistically better than the control group (Figure 2C). There was a small increase in the amount of slow wave sleep in the active group but no statistically significant changes in sleep architecture (Table 2).

3.4 Symptoms, Quality of Life, Neuropsychological Function and Blood Pressure

Sleepiness, mood, sleep apnea symptoms, quality of life, neuropsychological function and blood pressure improved in both groups post-treatment (Table 1). ANOVA revealed no significant difference in these measures between the two groups.

3.5 Relationship between supine sleep time and AHI

An exploratory analysis was conducted to examine the proportion of patients who exhibited a reduction in supine sleep and AHI within each group. Treatment efficacy was arbitrarily defined as a reduction in supine sleep to less than 10% of total sleep time; 64% active treatment subjects and

35% control group subjects achieved this. However this did not guarantee an AHI response – 15.9% active subjects and 18.9% control group subjects slept for less than 10% total sleep time on their back at the final PSG and yet had an AHI >10 (Table 3, Figure 4).

3.6 Predictors of Response

Pearson's correlation analysis revealed a significant association between the reduction in supine sleep as a proportion of total sleep time and the change in AHI with treatment in the group as a whole ($r = 0.298$, $p = 0.007$). The baseline variables which were different between responders and non-responders (i.e. post-treatment AHI<10 and post-treatment AHI $\geq$ 10 respectively) were AHI total ($p = 0.02$), AHI non-supine ($p = 0.01$), neck circumference ($p = 0.008$) and oxygen nadir ($p = 0.002$). In the first model, these four variables plus baseline supine sleep time (as % total sleep time) and group were entered into a binary logistic regression model, with the dependant categorical variable being AHI post-treatment <10 (Table 4). The significant predictors of a reduction in AHI to less than 10 were group (OR=5.57, p=0.003), oxygen nadir (OR=1.16, p=0.04) and AHI non-supine (OR=0.93, p=0.04). The variance explained by this model was $R^2 = 0.36$. In a second model, AHI total and group were replaced by an interaction term $AHI_{baseline}$ x group. In this model, the significant predictors of a fall in AHI to less than 10 were the interaction $AHI_{baseline}$ x group (OR=0.62, p=0.003) and nadir SaO_2 (OR=2.03, p=0.02), and the variance explained by this model (R^2) was 0.31 (Table 4).

4. DISCUSSION

This is the largest study to assess the therapeutic benefit of sleep position modification for the treatment of OSA. The simple and inexpensive position modification device used in the current study effectively prevented supine sleep, and reduced OSA severity in those with moderate and severe OSA (AHI ≥20). However, there was no impact of this treatment on neurobehavioral function, daytime sleepiness or blood pressure in the four week treatment period.

Several baseline factors predicted a fall in the AHI in these participants, including randomization to the active group, a lower non-supine AHI, having less oxygen desaturation, and an interaction effect with group allocation and total AHI. These factors accounted for approximately half the variance in the treatment response. It is not surprising that those with higher baseline total AHI and lower non-supine AHI should had the most benefit from this therapy. The more severe the disease, the more room there is for a treatment response and as such we would suggest that the definition of "positional OSA" should be reconsidered. If "positional OSA" was recast as referring to OSA which is characterised as having a non-supine AHI of < 10 and a supine AHI of at least double that (i.e. $\geq$20), then the definition would relate directly to treatment outcomes where treatment was directed at changing sleeping position. Future studies could explore this relationship in other cohorts to further test the validity of this assertion.

Apart from a reduction in the AHI, there was no benefit from using the position modification device on any of the clinical outcomes measured. It is likely that the sample size was insufficient to show an effect on the neuropsychological tests, but there was adequate estimated power to show an incremental benefit for daytime sleepiness, symptoms and quality of life, none of which were apparent. Several factors may have influenced this finding. The treatment time of 4 weeks may have not been adequate to show significant improvement in either the neurobehavioral or blood pressure outcomes. This lack of benefit seen may also be attributed to lack of adherence; although all of the participants reported usage of at least 6 hours per night with no side-effects, self-report adherence may be an over-estimate. Those in the control group were advised to sleep in a non-supine position and they also reduced both their amount of supine sleep and AHI. This would have reduced the likelihood of finding a difference in clinical outcomes between the groups.

The current study is in agreement with previous reports that the effect of the supine sleep position in increasing obstructive breathing events differs between obese and non-obese participants. We found that a smaller neck circumference indicated a greater likelihood of treatment response, supporting two previous studies.[4,35]

Many previous randomized controlled trials of the efficacy of sleep position modification for the treatment of OSA have been crossover studies, often comparing a position device to CPAP. The study of Jokic and colleagues used a split night PSG at baseline and full PSG for each efficacy study with no intervening washout period between the 2-week treatment periods. Thus, it is possible that there was inadequate evaluation of OSA severity and sleeping position at baseline. More recent studies[21, 22] used a portable unattended sleep monitor that recorded only respiratory channels and had a 1 week washout period between the treatment arms. Neither study was able to measure sleep architecture or arousals and none of the studies were adequately powered for sleepiness or quality of life outcomes. In our current larger, parallel study, the effect of usual conservative treatment on outcomes is clearly demonstrated, with participants in both arms having significant improvements in sleep disordered breathing, sleepiness, mood, quality of life, symptoms and neuropsychological outcomes. There was no impact of the device on sleep fragmentation or sleep architecture.

The choice of the control arm intervention is a potential limitation of the study; it is likely that there was a partial therapeutic effect of the sleep hygiene instructions. However such advice is routine in standard clinical practice so this comparison is particularly clinically relevant.

Major logistical problems facing sleep clinicians at the beginning of the 21st century include access to specialized diagnostic facilities[36] and the provision of well-tolerated and effective therapies that are not costly. In this study we have attempted to address the latter issue with a simple, inexpensive device in patients for whom sleep-disordered breathing is dependent upon body position during

sleep. In contrast to previous studies that found poor compliance with positional devices,[37] patients in the current study tolerated the positional device well. The current findings highlight the effectiveness of positional therapy, and the development and assessment of newer less invasive devices over longer time periods is clearly warranted.

5. Conclusions

This study has shown that a simple, inexpensive position device is more effective than giving patients advice about avoiding supine sleep in keeping patients with positional OSA in the non-supine sleeping position, and results in a reduction in AHI. This is an encouraging finding and supports the clinical impression that this type of thereapy is valuable for many patients. Longer duration studies of physical treatments that modify sleep position with greater participant numbers are needed to further explore whether additional clinical benefits in cardiovascular and metabolic risk, daytime sleepiness, quality of life and other neuropsychological parameters are achievable.[38] These studies should include objective measures of device adherence and sleep position during the period of the intervention. Our results suggest that those with a lower non-supine (< 10) and a higher supine (≥ 20) AHI are more likely to obtain a response to positional treatments. In addition to being used as a stand-alone treatment for position-dependent OSA, there may be an adjunct therapeutic role of sleep position modification in patients who have an inadequate response to CPAP or oral appliances or who can only tolerate treatment for a limited portion of the night. Evidence that such a simple inexpensive treatment is effective also enables improved access to OSA treatment for low income patients. Further evaluation of all these therapeutic areas is warranted.

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ABBREVIATIONS

AHI	Apnea Hypopnea Index
BMI	Body Mass Index
CPAP	Continuous Positive Airways Pressure
ESS	Epworth Sleepiness Scale
FOSQ	Functional Outcomes of Sleep Questionnaire
nonREM	non Rapid Eye Movement
OSA	Obstructive Sleep Apnea
PSG	Polysomnography
REM	Rapid Eye Movement
SASQ	Symptoms of Sleep Apnea Questionnaire
SD	Standard Deviation
TST	Total Sleep Time

Appendix One

TEN POINT GUIDE TO IMPROVING YOUR SLEEP APNEA WITH HEALTHY LIFESTYLE CHANGES

There are many simple changes in your day-to-day life that may improve your sleep apnea, reduce your snoring and make you feel less sleepy and more active during the day.

We recommend the following:

1. Lose weight

We recognise that this is not easy and you have probably tried many different diets.

Try following these simple rules:

- Write out a list of the foods that you are going to avoid – remember to be realistic. If you set your goals too high, failure is more likely. If going to the football and having a meat pie is an essential part of your life, then don't try to stop that.
- Stick to three good meals each day. Try not to snack between meals.
- Drink plenty of water
- Avoid anything that has been cooked in oil or butter
- Avoid processed foods such as salami and takeaway fast foods.
- Substitute margarine or butter in sandwiches for low fat cottage cheese.
- Have your main meal in the middle of the day, with a smaller meal in the evening.

2. Exercise

Exercise should be brisk walking, at least 5 times each week and for at least 30 minutes each time.

3. Sleep on your side

Studies have shown that sleep apnea events are improved by sleeping on your side. Try to sleep on your side whenever you are asleep.

4. Reduce alcohol intake

Alcohol is known to make sleep apnea worse, therefore avoid any alcohol for *at least 4 hours* before going to bed.

5. Get enough sleep

Everyone needs at least 8 hours sleep every night. You may think that you are performing well with less, but you will perform better during the day if you have the amount of sleep that your body really needs.

6. Establish a sleep routine

Let your body know when it is time to start "switching off" and getting ready for sleep. Have a calm routine for the half hour before you go to bed that you follow every night. For example, sit quietly and read for 15 minutes, brush teeth, check on the children, change into nightclothes, go to bed. Do not watch television in bed, your body should become accustomed to using bed only for sleeping.

You cannot expect to be partying one minute and be asleep the next.

7. Sleeping tablets

Try *not* to take any sleeping tablets or other sedatives, if you can possibly avoid them.

8. Nose Blockage

If your nose is blocked and you find it difficult to breathe through both sides, see your doctor for treatment advice.

9. Smoking

Nicotine is a stimulant, therefore you should avoid smoking for at least ½ hour before going to bed.

10. Caffeine

Caffeine is a stimulant, therefore you should avoid all caffeine-containing drinks and foods for 1 hour prior to going to bed. This includes tea, coffee and cola drinks.

FIGURE LEGENDS

Figure 1 Consort diagram of study flow

Figure 2 Means and confidence intervals of the amount of supine sleep as a percentage of total sleep time (A), apnea hypopnea index (B), and oxygen saturation nadir (C) at baseline and post-treatment in the active and control groups.

Figure 3 Individual Apnea Hypopnea indices for participants in the active (A) and the control (B) groups.

Figure 4 Scatterplot of the changes in AHI and change in sleeping position, in the active (white circles) and control (black squares) groups.

Table 1 - Baseline characteristics and response to treatment in the active and control groups

	Baseline		**After Treatment**	
	Active	Control	Active	Control
BMI (kg/m^2)	30.0 (5.3)	30.9 (7.7)	30.2 (5.3)	30.8 (7.7)
Total Sleep Time (mins)	346.7 (66.3)	326.0 (60.9)	334.0 (75.0)	352.6 (62.2)*
Time Supine (mins)**	130.9 (81.3)	158.6 (92.2)	28.4 (48.3)‡	86.4 (87.1)‡
Proportion of TST supine (%)**	37.7 (21.3)	48.7 (26.3)	8.7 (1.5)‡	24.0 (23.1)‡
AHI‖	20.1 (8.8)	21.8 (10.1)	10.8 (9.9)‡	16.8 (15.9)*
Supine AHI	43.2 (25.5)	39.7 (19.3)	35.5 (27.7)	37.9(25.5)
Non-Supine AHI	10.7 (9.0)	8.1 (6.1)	9.6 (10.0)	11.6 (13.8)
Arousal Index	23.7 (9.8)	17.9 (8.8)	14.2 (7.6)‡	13.8 (5.9)*
Minimum SaO_2 (%)	87.9 (4.1)	87.3 (5.0)	89.8 (4.4)†	87.9 (4.8)
Sleep Efficiency (%)	76.3 (12.0)	75.2 (12.9)	75.5 (15.5)	80.4 (12.7)
ESS	9.9 (4.7)	10.0 (5.9)	8.1 (4.1)‡	9.4 (6.6)
Symptom Score	58.5 (17.8)	58.5 (22.4)	47.0 (14.7)‡	54.4 (19.5)*
FOSQ Total Score	3.4 (0.4)	3.2 (0.6)	3.5 (0.4)†	3.3 (0.6)
Beck Depression Index	8.2 (5.1)	8.1 (5.5)	6.0 (4.9)‡	6.4 (4.9)†
POMS TMD	23.0 (23.0)	15.6 (24.4)	13.2 (17.6)‡	10.9 (15.5)

PVT f10	186.9 (37.0)	188.0 (15.7)	181.1 (15.8)	193.5 (30.9)
Neck circumference (cm)	40.6 (3.4)	40.8 (4.2)	40.6 (3.3)	40.9 (3.7)
		% participants		
Systolic BP (mmHg)	128.3 (15.5)	127.6 (15.4)	125.7 (9.6)	133.4 (15.2)
Diastolic BP (mmHg)	77.6 (9.2)	78.1 (10.6)	75.1 (9.2)*	79.4 (9.8)

All data are mean(SD)

BMI, body mass index; Minimum SaO_2, minimum oxygen saturation; ESS, Epworth Sleepiness Scale; FOSQ, Functional Outcomes of Sleep Questionnaire; pvt f10, Psychomotor Vigilance Task mean reaction time of fastest 10% responses; POMS TMD Profile of Mood States Total Mood Disorder.

* $p<0.05$, † $p<0.01$, ‡ $p<0.005$ Baseline vs. Treatment ANCOVA

ll $p<0.1$, **$p<0.05$ Post Treatment difference between groups ANCOVA

Table 2 Sleep Architecture as a percentage of total sleep time for the active and control groups at baseline and at follow-up

	Baseline		**After Treatment**	
	Active	Control	Active	Control
	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
Stage 1 (%)	11.4 (9.6)	9.0(7.3)	8.8 (6.6)	8.5 (9.1)
Stage 2 (%)	51.0 (11.1)	48.7 (13.5)	51.2 (9.9)	50.3 (13.5)
Stage 3/4 (%)	20.8 (11.4)	23.6 (15.2)	24.2 (9.0)	22.9 (14.8)
REM (%)	16.4 (6.0)	16.1 (8.1)	16.0 (6.6)	18.3 (4.8)

REM, rapid eye movement

Table 3 Distribution of AHI treatment response and supine sleep in the active and control groups at follow-up

Post Treatment		Active	Control
1	AHI≤10 + SupineTST≤10	45.5	16.2
2	AHI>10 + SupineTST≤10	15.9	18.9
3	AHI>10 + SupineTST>10	15.9	40.5
4	AHI≤10 + SupineTST>10	22.7	24.3

AHI, apnea-hypopnea index; Supine TST≤10, Supine sleep ≤ 10% total sleep time; SupineTST>10, Supine sleep > 10% total sleep time.

Table 4 Logistic regression of baseline predictors of AHI response

	B	SE	p	Odds Ratio
Model 1				
AHI NS	-0.09	0.04	0.028	0.52
Supine time (mins)	-0.11	0.04	0.004	0.38
Neck (cm)	-0.19	0.09	0.04	0.49
SaO_2 Nadir (%)	0.17	0.09	0.04	2.11
Group	2.19	0.71	0.002	8.92
Model 2				
AHI NS	-0.17	0.06	0.003	0.25
Supine time (mins)	-0.01	0.004	0.005	0.42
Neck (cm)	-0.20	0.09	0.03	0.47
SaO_2 Nadir (%)	0.18	0.08	0.04	2.19
AHIxgroup	0.13	0.04	0.001	3.14

AHI NS, Apnea Hypopnea Index non supine; SaO_2 nadir, oxygen saturation minimum; Group, active or conservative treatment group; AHIxGroup, interaction term for AHI total at baseline with treatment group.

Model 1: AHI nonsupine, supine time, neck size, minimum oxygen saturation and group (active or control)

Model 2: AHI nonsupine, supine time, neck size, minimum oxygen and interaction term for AHI total at baseline with treatment group.